AMENDMENTS TO THE SPECIFICATION

Please insert the following paragraph before paragraph 1 on page 1:

This application is a national phase entry of PCT application Serial No. JP2003/013958, filed October 29, 2003, which claims priority to and benefit of Japanese application No. 2002-315091, filed on October 29, 2002, each of which is incorporated herein by reference in their entireties.

Please amend the specification on page 1, paragraph 1 as follows:

The present invention relates to a non-human animal model that develops Guillain-Barré syndrome (Fisher syndrome), more specifically to a non-human animal model of Guillain-Barré syndrome which can be obtained by immunizing with ganglioside GQ1b a non-human animal model whose FcγRIIB-gene ~~function~~ is deficient in its chromosome (FcγRIIB-gene-deficient non-human animal model), and a screening method of a therapeutic agent for Guillain-Barré syndrome using the non-human animal model.

Please amend the paragraph on pages 6 (line 18) – page 7 (line 3) as follows:

The present invention relates to: a mouse model of Guillain-Barré syndrome which can be obtained by immunizing with gangliosides GQ1b a mouse whose FcγRIIB-gene ~~function~~ is deficient in its chromosome to develop Guillan-Barre syndrome ("1"); a mouse model of Guillain-Barré syndrome, wherein Guillain-Barré syndrome is Fisher syndrome ("2"); the mouse model of Guillain-Barré syndrome according to "1" or "2", which develops a peripheral neuropathy wherein paralysis of its tail and hind legs occurs ("3").

Please amend the paragraph on page 8 (lines 7-28) as follows:

As for the non-human animal model of Guillain-Barré syndrome of the present invention, there is no particular limitation as long as it is obtained by immunizing with ganglioside GQ1b the non-human animal whose FcγRIIB-gene ~~function~~ is deficient in its chromosome, and is a non-human animal that develops Guillan-Barré syndrome. Here Guillan-Barré syndrome indicates a non-hereditary disorder characterized by rapidly-progressing flaccid-motor paralysis (weakness in muscles of all four limbs), loss of deep tendon reflexes, dysphagia, articulatory disorder, deep sensory disturbance, and vegetative neurosis (cardiac arrhythmia, blood pressure fluctuation) which occurs a few weeks after a flu-like symptom, and other similar disorders. If the Guillan-Barré syndrome is developed, level of antibody titer against ganglioside GQ1b in the serum rises and more specifically, external ophthalmoplegia, diplopia, ataxia, loss of tendon reflexes, facial nerve palsy, peripheral neuropathy of tail and hind legs and the like are induced. Furthermore, Fisher syndrome is a variant of Guillan-Barré syndrome, whose symptoms are identical to those of Guillan-Barré syndrome except that quadriplegia is not induced in humans.

Please amend the paragraph on page 8 (line 29) - page 9 (line 27) as follows:

As for the FcγRIIB-gene-deficient non-human animal model of the present invention, any model animals are accepted as long as its FcγRIIB-gene ~~function~~ is deficient in its chromosome, although it can be preferably exemplified by rodents such as mice and rats, in particular, by the mouse whose FcγRIIB-gene ~~function~~ is deficient in its chromosome. The mouse whose FcγRIIB-gene ~~function~~ is deficient in its chromosome can be generated according to the method previously described by the present inventors (Nature 379, 346-349, 1996) and the

like. In concrete terms, FcγRIIB-knockout mouse can be obtained according to the following process: FcγRIIB gene is screened using a gene fragment derived from the mouse gene library by a method such as PCR and the like; the screened FcγRIIB gene is subcloned using a viral vector and the like, then determined by DNA sequencing; a target vector is prepared by substituting the fragment containing S_2 exon and EC_1 exon of the clone to a pMC1 neo gene cassette and the like; the linearized vector is introduced into ES cells by electroporation and the like to cause homologous recombination; from among the homologous recombinants, ES cells showing resistance to G418 and the like are selected, and the clones of those cells are microinjected into a murine blastocyst; the blastocyst is placed back to the host parent to generate a chimeric mouse; when this chimeric mouse is intercrossed with a wild-type mouse, a heterozygous mouse can be obtained; by intercrossing the heterozygous mice, an FcγRIIB-knockout mouse can be obtained.